UNITED STATES BANKRUPTCY COURT
DISTRICT OF DELAWARE

IN RE:	.	Case No. 01-1139 (JKF)
	.	
W.R. GRACE & CO.,	.	
et al.,	.	USX Tower - 54th Floor
	.	600 Grant Street
	.	Pittsburgh, PA 15219
Debtors.	.	
	.	April 1, 2008
. . . .	.	9:08 a.m.

TRANSCRIPT OF TRIAL
BEFORE HONORABLE JUDITH K. FITZGERALD
UNITED STATES BANKRUPTCY COURT JUDGE

APPEARANCES:

For the Debtors: Kirkland & Ellis, LLP
By: DAVID BERNICK, ESQ.
BARBARA HARDING, ESQ.
JANET BAER, ESQ.
BRIAN STANSBURY, ESQ.
SAL BIANCA, ESQ.
RAINA JONES, ESQ.
HENRY THOMPSON, ESQ.
SCOTT McMILLAN, ESQ.
200 East Randolph Drive
Chicago, IL 60601

Kirkland & Ellis, LLP
By: THEODORE FREEDMAN, ESQ.
Citigroup Center, 153 East 53rd St.
New York, NY 10022

Audio Operator: Janet Heller

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J&J COURT TRANSCRIBERS, INC.
268 Evergreen Avenue
Hamilton, New Jersey 08619
E-mail: jjcourt@optonline.net

(609)586-2311 Fax No. (609) 587-3599

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APPEARANCES (CONT'D):

For the Asbestos Creditors Committee:	Caplin & Drysdale, Chartered By: BERNARD BAILOR, ESQ. PETER LOCKWOOD, ESQ. NATHAN FINCH, ESQ. One Thomas Circle, NW Washington, D.C. 20005
	Caplin & Drysdale, Chartered By: ELIHU INSELBUCH, ESQ. 375 Park Avenue, #3505 New York, NY 10152
For the Debtors:	ARPC By: AMY BROCKMAN, ESQ.
For W.R. Grace:	W.R. Grace By: MARK SHELNITZ, ESQ. JAY HUGHES, ESQ. WILLIAM CORCORAN, ESQ. 7500 Grace Drive Columbia, MD 21044
For the Equity Committee:	Kramer Levin Naftalis & Frankel By: GREGORY HOROWITZ, ESQ. 919 Third Avenue New York, NY 10022
For the Unsecured Creditors' Committee:	Stroock & Stroock & Lavan By: KENNETH PASQUALE, ESQ. ARLENE KRIEGER, ESQ. 180 Maiden Lane New York, NY 10038-4982
For the Property Damage Committee:	Bilzin Sumberg Baena Price & Axelrod LLP By: MATTHEW KRAMER, ESQ. SCOTT BAENA, ESQ. JAY SAKALO, ESQ. 200 South Biscayne Boulevard Suite 2500 Miami, FL 33131

APPEARANCES (CONT'D):

For the Ad Hoc Committee of Equity Sec. Holders:	Dewey & LeBoeuf, LLP By: JENNIFER WHITENER, ESQ. 125 West 55th Street New York, NY 10019
For the Future Claimants Representatives:	Orrick, Herrington & Sutcliffe LLP By: ROGER FRANKEL, ESQ. ANTHONY KIM, ESQ. RAYMOND MULLADY, ESQ. JOHN ANSBRO, ESQ. Washington Harbour 3050 K Street, N.W. Washington, D.C. 20007
For Committee of Asbestos Personal Injury Claimants:	Campbell & Levine By: MARK T. HURFORD, ESQ. 800 North King Street Suite 300 Wilmington, DE 19701
For Maryland Casualty:	Connelly Bove Lodge & Hutz, LLP By: JEFFREY WISLER, ESQ. The Nemours Building 1007 North Orange Street Wilmington, DE 19899
For Maryland Casualty:	Eckert Seamans Cherin & Mellott, LLC By: EDWARD LONGOSZ, II, ESQ. 1747 Pennsylvania Avenue, N.W. Suite 1200 Washington, D.C. 20006
	STB By: STERLING MARSHALL, ESQ.
For Sealed Air:	Skadden, Arps, Slate, Meagher & Flom, LLP By: DAVID TURETSKY, ESQ. One Rodney Square Wilmington, DE 19801

APPEARANCES (CONT'D):

For Sealed Air:	NERA Economic Consulting By: STEPHANIE PLANCICH 1166 Avenue of the Americas 28th Floor New York, NY 10036
For W.R. Grace:	NERA By: ELENA ZAPRYANOVA LINDA SHEN
For Serengeti:	Vinson & Elkins, LLP By: ARI BERMAN, ESQ. Trammell Crow Center 2001 Ross Avenue, Suite 3700 Dallas, TX 75201 By: BILLAL SIKANDER
For Silver Point Capital:	Silver Point Capital By: JOHN KU
For the Debtors:	Pachulski, Stang, Ziehl &Jones By: JAMES O'NEILL, ESQ. 919 North Market Street 17th Floor Wilmington, DE 19899-8705

TELEPHONIC APPEARANCES:

For the Unsecured Creditors' Committee:	Strook & Strook & Lavan By: LEWIS KRUGER, ESQ. 180 Maiden Lane New York, NY 10038
For Ad Hoc Committee:	Weil, Gotshal & Manges By: M. JARRAD WRIGHT, ESQ. 1300 Eye Street NW, Suite 900 Washington, D.C. 20005
For Official Committee of Equity Holders:	Kramer Levin Naftalis & Frankel LLP By: PHILLIP BENTLEY, ESQ. 919 Third Avenue New York, NY 10022

TELEPHONIC APPEARANCES (CONT'D):

For Official Committee of Asbestos Property Damage Claimants:	Dies & Hile, LLP By: MARTIN DIES, ESQ. 1601 Rio Grande, Suite 330 Austin, TX 78701 ELIZABETH DEVINE, ESQ.
For Various Claimant Firms:	Stutzman, Bromberg, Esserman & Plifka By: DAVID J. PARSONS, ESQ. VAN J. HOOKER, ESQ. SANDER L. ESSERMAN, ESQ. 2323 Bryan Street Suite 2200 Dallas, TX 75201
For Fireman's Fund:	Stevens & Lee, P.C. By: JOHN DEMMY, ESQ. DAVID R. BEANE, ESQ. 1105 North Market Street, 7th Fl. Wilmington, DE 19801
For Owens-Illinois:	McCarter & English By: DANIEL SILVER, ESQ. Renaissance Centre, 405 N. King St. Wilmington, DE 19801
For David T. Austern:	Piper Jaffray & Co. By: JONATHAN BROWNSTEIN, ESQ.
For Asbestos Property Damage Claimants:	Scott Law Group By: DARRELL SCOTT, ESQ. 1001 East Main Street, Suite 500 Sevierville, TN 37864
For National Union Fire Insurance Co.:	Zeichner Ellman & Krause, LLP By: MATTHEW RUSSELL, ESQ. ROBERT GUTTMANN, ESQ. MICHAEL DAVIS, ESQ. 575 Lexington Avenue New York, NY 10022

TELEPHONIC APPEARANCES (CONT'D):

For the Future Claimants Representatives:	Orrick, Herrington & Sutcliffe, LLP By: DEBRA FELDER, ESQ. JOSHUA CUTLER, ESQ. Washington Harbour 3050 K Street, N.W. Washington, D.C. 20007 For
For Federal Insurance Company:	Cozen O'Connor By: JEFFREY WAXMAN, ESQ. Chase Manhattan Centre 1201 North Market Street Wilmington, DE 19801
For Federal Insurance Company:	Cozen O'Connor By: JACOB C. COHN, ESQ. 1900 Market Street Philadelphia, PA 19103
For Allstate Insurance:	Cuyler Burk, LLP By: ANDREW CRAIG, ESQ. Parsippany Corporate Center Four Century Drive Parsippany, NJ 07054
For W.R. Grace:	W.R. Grace By: WILLIAM CORCORAN, ESQ. 7500 Grace Drive Columbia, MD 21044
	Kirkland & Ellis, LLP By: ELLEN AHERN, ESQ. 200 East Randolph Drive Chicago, IL 60601
	Kirkland & Ellis, LLP By: DAVID MENDELSON, ESQ. 6555 Fifteenth Street, N.W. Washington, DC 20005
For State of Montana Department of Environmental Quality:	Womble Carlyle Sandridge & Rice By: FRANCIS MONACO, ESQ. 222 Delaware Avenue Suite 1501 Wilmington, DE 19801

TELEPHONIC APPEARANCES (CONT'D):

For Official Committee of Asbestos Personal Injury Claimants:	Anderson Kill & Olick By: ROBERT M. HORKOVICH, ESQ. 1251 Avenue of the Americas New York, NY 10020-1186
For W.R. Grace:	Cohn Whitesell & Goldberg, LLP By: NATHAN SOUCY, ESQ. 101 Arch Street Boston, MA 02110
For CNA:	Goodwin Procter, LLP By: DANIEL GLOSBAND, ESQ. Exchange Place Boston, MA 02109-2881
For Grace Certain Cancer Claimants:	Montgomery, McCracken, Walker & Rhoads, LLP By: NATALIE D. RAMSEY, ESQ. 300 Delaware Avenue, Ste. 750 Wilmington, DE 19801
For David T. Austern, the Future Claimants' Representative:	Phillips, Goldman & Spence, P.A. By: JOHN C. PHILLIPS, ESQ. 1200 North Broom Street Wilmington, DE 19806
For W.R. Grace:	Pachulski, Stang, Ziehl & Jones, LLP By: TIMOTHY P. CAIRNS, ESQ. 919 North Market Street 17th Floor Wilmington, DE 19899-8705
For the Asbestos Creditors Committee:	Caplin & Drysdale, Chartered By: WALTER SLOCOMBE, ESQ. JEANNA RICKARDS, ESQ. JAMES WEHNER, ESQ. LESLIE KELLEHER, ESQ. One Thomas Circle, NW Washington, D.C. 20005
For the Asbestos Creditors Committee:	Ferry Joseph & Pearce, P.A. By: THEODORE TACCONELLI, ESQ. 824 Market Street, Suite 19899 Wilmington, DE 19899

TELEPHONIC APPEARANCES (CONT'D):

For Ford, Marrin, Esposito, Witmeyer & Gleser:	Ford, Marrin, Esposito, Witmeyer & Gleser By: SHAYNE SPENCER, ESQ. Wall Street Plaza New York, NY 10005
For Pepsi:	Butler Rubin Salfarelli & Boyd, LLP By: KIRK T. HARTLEY, ESQ. 70 West Madison Street Suite 1800 Chicago, IL 60602
For Official Committee of Unsecured Creditors:	Duane Morris, LLP By: MICHAEL LASTOWSKI, ESQ. 1100 North Market Street, Suite 1200 Wilmington, DE 19801-1246
For Official Committee of Asbestos Property Damage Claimants:	Brandi Law Firm By: TERENCE D. EDWARDS, ESQ. 44 Montgomery St., Suite 1050 San Francisco, CA 94104
For the State of CA, Dept. of Gen. Services:	Hahn & Hessen, LLP By: CHRISTINA J. KANG, ESQ. 488 Madison Avenue, 14th Fl. New York, NY 10022
For Baron & Budd, et al.:	Hogan Firm Attorneys at Law By: DANIEL K. HOGAN, ESQ. 1311 Delaware Avenue Wilmington, DE 19801
For the PD Committee:	Speights & Runyan By: DANIEL SPEIGHTS, ESQ. 200 Jackson Avenue, East Hampton, SC 29924
For Royal Insurance:	Wilson Elser Moskowitz Edelman & Dicker, LLP By: CATHERINE CHEN, ESQ. 150 East 42nd Street New York, NY 10017
For David T. Austern:	Piper Jaffray & Co. By: JASON SOLGANICK

TELEPHONIC APPEARANCES (CONT'D):

For Scott Company:	Vorys, Sater, Seymour & Pease, LLP By: TIFFANY COBB, ESQ. 52 East Gay Street Columbus, OH 43216
For London Market Companies:	Mendes & Mount, LLP By: ALEXANDER MUELLER, ESQ. 750 Seventh Avenue New York, NY 10019-6829
For Official Committee of Asbestos Property Claimants:	LECG By: ALAN MADIAN, ESQ.
For Official Committee of Asbestos Property Claimants:	Richardson Patrick Westbrook & Brickman, P.C. By: EDWARD J. WESTBROOK, ESQ. 174 East Bay Street Charleston, SC 29401
For Ivory Investment:	Ivory Investment By: DHANANJAY PATWARDHAN
For Linden Advisors:	Linden Advisors, LP By: CRAIG GILBERT
For O'Conner:	O'Conner By: John R. Wollen
For Credit Suisse First Boston:	Credit Suisse First Boston By: TIM McARDLE
For King Street Capital Management, LLC:	King Street Capital Management, LLC By: KIM CHRISTENSEN, ESQ.
For the Blackstone Group:	The Blackstone Group By: JOHN O'CONNELL
For Dune Capital Mgmt:	Dune Capital Management By: GUY BARON

TELEPHONIC APPEARANCES (CONT'D):

For Anchorage Advisors:	Anchorage Advisors By: JONATHAN LEWINSOHN
For Lehman Brothers:	Lehman Brothers By: ANDREW CHAN
For Caxton Associates:	Caxton Associates, LLC By: JAMES RIEGER
For Dow Jones News Wires:	Dow Jones News Wires By: PEG BRICKLEY
For Citadel Investment Group:	Citadel Investment Group By: BEAU HARBOUR
For Durham Asset Management:	Durham Asset Management By: JEFFREY A. ROSENKRANZ
For Murray Capital Management	Murray Capital Management, Inc. By: MARTI MURRAY
For Korn Capital, LLC:	Korn Capital, LLC By: STEPHANIE KWONG
For Irwin H. Zandman:	Irwin H. Zandman By: IRWIN H. ZANDMAN
For Venor Capital:	MICHAEL SCOTT, ESQ. Washington, DC
For Asbestos Claimants:	Brayton Purcell, LLP By: CHRISTINA SKUBIC, ESQ. 222 Rush Landing Road Novato, CA 94948
For Halycon Asset Management LLC:	Halcyon Asset Management, LLC By: JOHN GREENE

TELEPHONIC APPEARANCES (CONT'D)

For The Scotts Co.:	Vorys, Sater, Seymour and Pease, LLP By: MATTHEW DAIKER, ESQ. 52 East Gay Street P.O. Box 1008 Columbus, OH 43216
For Private Investors:	WILLIAM M. WAGNER
For Bank of New York:	ANDREW HAIN, ESQ.
For WR Grace Shareholder:	Tocqueville Asset Management By: PETER SHAWN
For Christopher M. Candon:	Cohn Whitesell & Goldberg, LLP By: CHRISTOPHER M. CANDON, ESQ. 101 Arch Street Boston, MA 02110
For Deutsche Bank:	MATT DOHENY
DebtWire:	SETH BURNDY
For CNA Insurance:	Goodwin Proctor, LLP By: BRIAN MUKHERJEE, ESQ. Exchange Place 53 State Street Boston, MA 02109
For In Propra Persona:	Old Lane Law Office By: Rajeev Narang, Esq.

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THE COURT: Please be seated. Folks, I know it's very hot in here. I started complaining about it yesterday. Apparently it's a building-wide problem that they're looking into that I can't do anything about. I've asked people to try to go purchase some fans that may help in the meantime. I strongly suggest you take off your jackets, because otherwise it's going to be very uncomfortable and I may divest myself of my robe at some point. So, yes, I know, that's really scary, but nonetheless it may happen because it really gets unbearably hot until the building figures something out. So, please, I don't want anybody passing out from heat stroke. So, please, make yourselves comfortable.

Oh, I guess I should call the case, first.

MR. BERNICK: Yes, we should probably do that.

THE COURT: All right. This is the continuation of the personal injury estimation trial on W.R. Grace, 01-1139.

Lucky participants by phone today, I suppose, James Rieger, James O'Neill, Kim Christiansen, Ari Berman, Matt Doheny, Shayne Spencer, Alex Mueller, Francis Monaco, Robert Guttmann, James Wehner, Walter Slocombe, Jeanna Rickards, Peter Lockwood, Mark Hurford, Leslie Kelleher, Michael Davis, Bernard Bailor, Christina Kang, Michael Lastowski, Seth Brumby, Matthew Daiker, Daniel Silver, Catherine Chen, Debra Felder, Marti Murray, Christopher Candon, John Phillips, John Ku, David Turetsky, Robert Horkovich, Matthew Kramer, Theodore

Tacconelli, Lewis Kruger, Natalie Ramsey, Jonathan Brownstein, Jason Solganick, Janet Baer, Darrell Scott, David Beane, Elizabeth Devine, Alan Madian, Martin Dies, Terrance Edwards, Christina Skubic, Peter Shawn, Edward Westbrook, Scott Baena, Jay Sakalo, Rajeev Narang, Beau Harbour, Matthew Russell, David Parsons, Brian Mukherjee, Guy Baron, Andrew Chan, Daniel Speights and Andrew Craig. I'll take entries in court, please, give me one second here. Okay, thank you.

MR. BERNICK: David Bernick for Grace.

MS. HARDING: Barbara Harding for Grace.

MR. McMILLAN: Scott McMillan for Grace.

MR. HOROWITZ: Greg Horowitz for the Equity Committee.

MR. PASQUALE: Ken Pasquale for the Unsecured Creditors Committee.

MR. FINCH: Nathan Finch for the Asbestos Claimants Committee.

MR. BAILOR: Bernard Bailor for the Asbestos Claimants Committee.

MR. INSELBUCH: Elihu Inselbuch for the Asbestos Creditors Committee.

MR. MULLADY: Ray Mullady for the FCR.

MR. ANSBRO: Good morning, Your Honor, John Ansbro for the FCR.

MR. FRANKEL: Good morning, Your Honor, Roger Frankel

for the FCR.

THE COURT: Mr. Finch.

MR. FINCH: Yes. Before we proceed with Dr. Brody's testimony, I just want to make one statement for the record with respect to ACC-34. That's the document from the Halpain file, related to the Halpain file.

THE COURT: Yes.

MR. FINCH: The Halpain file itself is in evidence as ACC-472. It was one of the files that Dr. Florence testified was selected to be reviewed by the Celotex trust and by Exponent.

That document, ACC-472 clearly shows that materials were withheld on the last page of the document and the two pages from the back. ACC Exhibit 34 is clearly the materials that were withheld. ACC Exhibit 34 contains information that is relevant to Mr. Halpain's work with asbestos and specifically asbestos Zonolite and whether he was a mixer or not. We can argue later what, if any, inferences should be drawn from that and the Court will decide that. But, the arguments do not affect the relevance of the document. That's the position I want to be placed on the record with respect to that document.

THE COURT: All right. Mr. Bernick.

MR. BERNICK: If that was -- I'm sorry, if the purpose of that was to make a statement on the record, I

believe it's identical to the same statement that was made about 45 times yesterday. And I don't think that that really has changed the picture. They argue that it's relevance, the question is what's the foundation for the document, or the basis on which relevance can be assessed. So, Mr. Finch has made a statement about the providence of the document and the like, I had no notice of the fact this morning this would be raised yet again. We are in the process of tracking down something of the history of what happened to that document and I'm not prepared to address it today. But, I think the Court already has ruled, repeatedly, that there has to be further foundation before that document can come in. It's been proffered, that's the state of the record and at the time I'm in a position to respond with more information, I'll be anxious to do so.

MR. FINCH: With respect to the foundation, I think there's no dispute, Your Honor, the document is authentic and there's no dispute that ACC-34 is referenced, or at least the letters are referenced in the last page of the Halpain file. That's the foundational argument.

THE COURT: I don't see a foundation issue. It seems that the letters are referenced in the last page of the document, I think the question is, from the perspective of whether or not the Zonolite was actually an asbestos containing product because there were Zonolite products that were not

asbestos containing and the issue, I thought, was whether or not the document was not considered in the process of review because of the asbestos containing nature of the product, and the validity of the information in the document itself, and we don't have the witness here who actually excluded that consideration. We simply don't know why it was not considered.

MR. FINCH: Okay, well, the point is, it wasn't considered, Your Honor, and whatever inferences can be drawn from that document are there to be drawn.

THE COURT: I think Mr. Bernick conceded that for purposes of saying that the information was not considered, he has no problem with agreeing that the information was not considered. He made that agreement yesterday and I also agree that the information wasn't considered. Whatever the information was, it wasn't considered. That's clear.

If that's the purpose for the introduction of the document, to say here's a document that wasn't considered, okay, but the truth of the matter within the document is another issue, and as to that, I sustain the objection, right now. There is no foundation for me with respect to the truth.

MR. FINCH: Well, for the purposes of the document wasn't considered and what the document says on its face, we do offer Exhibit 34.

THE COURT: But that's the problem, because what the document says on its face and why it wasn't considered, you

need a foundation for. The fact that it wasn't considered is clear, everyone agrees to that, but why it wasn't considered, you need a witness to explain.

MR. FINCH: Your Honor, what the document says is also clear. There's no dispute about what the document says and there's also no dispute about what Exponent's procedures were. Ms. Anderson testified to that.

THE COURT: That's right.

MR. FINCH: Whatever inferences can be drawn from that or not drawn from that are already -- the facts establishing that are in the record. We would offer Exhibit 34 for the purposes that I stated.

THE COURT: You did offer it yesterday.

MR. FINCH: Yes.

THE COURT: Okay. And I said that subject to your connecting it up, I would withhold the rulings. You have the opportunity to get a witness here to explain why it was withheld or why it was not considered. That's what I mean to say.

MR. FINCH: All right. I offer it at this time, Your Honor. Are you sustaining the objection with respect to it?

THE COURT: I am making the same ruling that I made yesterday. You've given me nothing new. I agree that the information was not considered, in order to tell me why it was not considered and to have any inferences drawn from that, you

need to lay a foundation.

MR. FINCH: We think an adequate foundation has been laid as to what the document says and that it was not considered and that there are inferences that can be drawn from those two facts and, therefore, we offer it for that purpose.

THE COURT: All right.

MR. BERNICK: Your Honor, this really, I think, is not appropriate. We are now spending yet more time on a matter as to which there was a --

MR. FINCH: I --

MR. BERNICK: Excuse me. There's a dirth of information yesterday, there's a dirth of information today.

COURT CLERK: Can you move closer to the microphone.

MR. BERNICK: There's a dirth of information today. I've made a representation to the Court about what we're prepared to do. While Mr. Inselbuch and Mr. Finch have a conversation --

THE COURT: Pardon me, gentlemen, you can't talk over each other.

MR. BERNICK: -- about the instructions for the day, I would like to get on with the witness who we were told was only available this morning, and get it done. And if they then want to argue about this some more, they can argue about it some more. I would think that it's relatively simple. As Your Honor has indicated, we agree that the document was not

considered. The circumstances surrounding that are unclear. The probative value of that document already has been addressed in part by Dr. Florence from the stand, as if the document was not considered and should have been considered and that is that he had it before him. So, that amount of testimony has been taken.

With respect to what the document actually says, which is the purpose of the proffer, not as to whether it should have been considered but what the document actually says, there's nothing further we can do because we have no witness who can pin down exactly what that product was, for that matter, exactly what the deposition said. That was a summary by a lawyer, of a deposition. We don't have the deposition before the Court. So there are a variety of facts that are out there and we are wasting time.

THE COURT: All right. I've already ruled. To the extent that the offer is that the document was not considered, I accept the offer for that purpose and that purpose only as to why, you need a foundation and to make a connection, and the deposition was not offered and is not part of the record. Exhibit 34, however, is the offer and I've made the rulings with respect to that.

Let me make a note so I know what --

MR. FINCH: One final statement, Your Honor, for the record. The document on its face, 472, says privileged

document information and one can draw the conclusion from that that it was pulled out of the file on privilege grounds.

MR. BERNICK: Of course, it can be and, of course, the inference that Mr. Inselbuch is anxious that Mr. Finch draw to the Court is that there's some kind of, you know, secreting of documents, so that they're not divulged and that's exactly the kind of inference that we object to. This is just an effort to try to create some coloration here, without foundation. The process of putting these files together, Your Honor, was a process that they initiated because they wanted to conduct discovery with respect to lawyers files. And they were permitted to conduct that discovery, indeed, with our agreement as to non-waiver. So, they got those documents, they got them actually a long time ago and the question of whether they connected up with this particular file was apparently something that didn't even dawn on the ACC and the FCR until sometime within the recent past, otherwise, presumably, they would have brought it to the attention of the Court in connection with the expert reports.

So, there's a long deep history of who knew what when with respect to this document. I've instructed our people to put together that history so it's fully before the Court. Until such time as that occurs and there are facts that are known and are ascertainable, I believe it is inappropriate for counsel to make these veiled suggestions, that in some fashion

the privilege has been improperly asserted, it was not improperly asserted or improperly maintained. It was not improperly maintained, indeed, it was specifically preserved when those files were produced over our 408 objection.

So, all this stuff, all this -- what we're sitting here talking about this morning is an attempt without foundation to make some color in this record, instead of getting on with the evidence.

MR. FINCH: Your Honor, we've made our position clear, we've offered the document and my understanding is, you're not accepting the document other than to -- for the purposes stated on the record. At this time, the ACC --

THE COURT: And I'm not accepting a re-offer. I made rulings yesterday. I don't understand the process by which a Court makes rulings and then you come back into court and you reargue the same rulings that the Court made yesterday. I mean, I'm not an appellate court for my own rulings. So, this isn't a proper trial procedure either, Mr. Finch.

MR. FINCH: Thank you, Your Honor. At this time, with the consent of the debtors, the Asbestos Claimants Committee calls Dr. Arnold Brody, and my partner Bernard Bailor will be presenting that witness.

MR. BERNICK: The debtor Grace has consented to calling Mr. -- Dr. Brody, excuse me, out of order, to accommodate his schedule. We are not resting and I'm assuming

that there's an agreement that by his being called out of order, we're not waiving any kinds of rights as to the admissibility of the evidence or with respect to whether his testimony is within the scope, or whether our case can be complete in the fashion that was originally intended to be completed.

MR. FINCH: That's agreed, Your Honor. They're not resting and we're not asking them to waive any of their rights.

THE COURT: All right.

MR. MULLADY: Also agreed to by the FCR.

THE COURT: All right, thank you. Dr. Brody. If you'd be sworn, sir, please.

COURT CLERK: Please raise your right hand.

DR. ARNOLD BRODY, ACC WITNESS, SWORN

DIRECT EXAMINATION

BY MR. BAILOR:

Q Good morning, doctor.

A Good morning.

Q Will you please state your full name for the record.

A It is Arnold R. Brody, B-r-o-d-y.

Q And what is your present address?

A It's the North Carolina State University, the Department of Molecular Biomedical Sciences.

MR. BAILOR: Your Honor, we are offering Dr. Brody as our initial witness. He will explain to the Court the disease

processes related to asbestos, which will serve as a foundation for later testimony.

I would also note for the Court that some of his testimony will be somewhat technical in nature and we invite the Court, at any time, to interrupt for any further explanations if things get a little too technical.

THE COURT: All right, thank you.

Q Dr. Brody, what is your present position at North Carolina State University?

A I'm a professor as I say, in the Department of Molecular and Biomedical Sciences.

Q And what does the Department of Molecular and Biomedical Science do?

A Well, we have, mostly basic science research. We're carrying out work that's supported by the National Institutes of Health to understand a whole variety of different sorts of diseases.

Q And how long have you been at North Carolina State University?

A About a year and a half now.

MR. BAILOR: May I approach, Your Honor?

THE COURT: Yes, sir.

THE WITNESS: I think I've got these, Bernie.

MR. BAILOR: This is updated.

THE WITNESS: Oh, okay. Updated.

THE COURT: Mr. Bailor, I think I'm going to need one, too.

MR. BAILOR: Oh, I'm sorry.

THE WITNESS: You can have this one, I think I know what's in it.

THE COURT: That's okay, sir. They may want you to refer to it.

MR. BERNICK: I don't know if that's the updated one or not.

MR. BAILOR: I'm sorry, Your Honor.

THE COURT: That's all right, thank you.

Q Dr. Brody, I've handed you a document that's marked AC/FCR, I believe 274, is that correct?

A 271.

Q I'm sorry, 271. Could you please identify that for the Court?

A Yes. That's my current curriculum vitae.

Q Okay. Could we have --

THE COURT: Mr. Bailor, excuse me.

(Pause)

MR. BAILOR: Could we have ACC/FCR-835, first page?

Q Dr. Brody, could you explain to the Court your educational background?

A Sure. Starting at the bottom of that particular slide, you can see actually, it's a -- I don't know where you got the

BA, but I got a BS, which is a Bachelor of Science degree at Colorado State and that was in Zoology, which is the study of animals. Then I went to the University of Illinois where I received a Master of Science degree and that was in vertebrate anatomy, that includes animal and human anatomy, which is how our parts all fit together and how they function.

Then I went back to Colorado to do a PhD, doctorate in cell biology and every living thing is made of cells, we need to understand how cells function. Every disease has a target cell from which that disease develops and cell biologists, like myself study cell biology in that context.

The big word there "ultra-structural cytology" means the study of cell using what's called an electron microscope. This is a kind of microscope that magnifies things hundreds of thousands of times and that's the research tool that I was trained in at Colorado State.

Q Could we have the next page, please. Could you describe for the Court your professional experience?

A Right. So, actually, after I did my PhD, I did three years of post-doctoral study at Ohio State University. That's not on there, but a post-doctoral fellowship means that's where one finds out if their temperament and their intelligence is suited for a research laboratory. That worked out fine and I went to the University of Vermont.

So, my first professional assignment, my first

academic position was as an assistant professor at the University of Vermont and that would appear down at the bottom of that slide. I was there for six years and then I went to the National Institute of Environmental Health Sciences and that's what appears at the bottom there, and that was a position with the federal government and I was the head of the lone pathology laboratory there for, as you can see, for 15 years. Rose through the ranks to a level of what was called professor, within the National Institutes of Health

I then, in 1993, received an offer to go to the Pathology Department at Tulane University Medical Center in New Orleans. I was there starting in 1993 and then in 1999 I was promoted to the vice chairman of the department. So, I was the vice chairman of the Pathology Department in the Medical School at Tulane University from 1999 to 2005, and I say 2005 because that's when Hurricane Katrina came through. And Hurricane Katrina blew a lot of things out of the city including us. And it was in 2006, then, that I went to North Carolina State University and accepted a professorship there which is where I currently am doing my work.

MR. BAILOR: Can we have the next slide, please?

Q The next slide indicates some other positions you have held. Could you please describe these to the Court?

A Well, the top one, that's when I was at the National Institutes of Health, that is the position that I held and that

was the head of that group, and pathology is the study of disease, and, of course, pulmonary means the lungs and that's where my focus has been.

When I was at Tulane University, I organized what's called a lung biology program, and that was in the Center for Bio-Environmental Research.

MR. BAILOR: Can we have the next page, please? Q Could you explain your editorial positions?

A So, this is a list of what are called biomedical journals. These are journals that I publish in and these are journals where I've been asked to sit on the editorial boards to review the work of others. So, whenever I send my work in for publication, it, of course, is reviewed by a series of scientists and editors and by the same token I'm asked to serve in that review capacity as well and this is the list of some of the journals on which I serve on the editorial boards.

Q Now, Dr. Brody, have you done work specifically related to asbestos and its affects on the human body?

A Yes, certainly. When I was at the University of Vermont, we had a visitor to the department and his name was Dr. Wagner, Chris Wagner, it's W-a-g-n-e-r. I don't know if you've heard about Dr. Wagner yet in this court, but -- have you, actually?

Q No, we have not, doctor.

A Okay. So, Dr. Wagner discovered in 1960 that asbestos causes mesothelioma, this cancer that we've heard so much

about. Dr. Wagner saw the work that I was doing, using an electron microscope and he invited me to come and work with him in Wales, in the United Kingdom. This was a great opportunity for my career and for my family, to go and visit and work with Dr. Wagner. And he showed me at that time that all of the asbestos varieties cause all of the diseases; asbestosis, lung cancer, mesothelioma. And he had developed an animal model. He showed that you could use rats to understand human disease and this, of course, scientists across the world use animal models, but this was my first opportunity to use an animal model to understand human disease.

So, when I went back to the National Institutes of Health, that's where I started my own research program using these animal models to understand human disease, based on a lot of what I had learned personally from Dr. Wagner.

Q All right. Have you published in the area of asbestos and disease?

A Yeah, sure. If you look at my CV I list 146 peer-reviewed papers and 50 book chapters, and of the 146 peer-reviewed papers, probably 100 or so of them deal directly with asbestos. The others relate to lung disease and diseases caused by other agents, other than asbestos. The 50 book chapters are almost entirely related to asbestos and how asbestos causes disease.

Q Have you previously testified as an expert in court with respect to asbestos-related diseases?

A Many times. Many times, sure.

Q Can you give us an estimate?

A Well, I had one case in 1989, and I had a few cases in the years thereafter, from '96 or '97 or so, I've been testifying mostly for plaintiffs, I would say about once or twice a month, since '96/'97. As I say, mostly for plaintiffs, but I've testified for a number of manufacturers, as well. I heard the word Celotex in court, I testified for the Celotex Corporation in their bankruptcy hearings in Florida. I've testified for a number of other asbestos manufacturers as well, over the years, but typically I testify for plaintiffs.

But my testimony is how asbestos causes disease, of course.

MR. BAILOR: Your Honor, at this point we would tender Dr. Brody as an expert in cellular and molecular biology and how asbestos affects the human body on a cellular and on a molecular level.

MR. BERNICK: No objection.

THE COURT: The witness is accepted as an expert in that field.

Q Dr. Brody, have you prepared a series of slides that will help you explain how asbestos affects the human body?

A I have a series of slides that I've used in many courtrooms before. They are slides, most of them are pictures that I've taken with various microscopes over the years. I

have used many of these slides in medical school lectures and various universities where I've lectured in this country and around the world, and I put together a group for this courtroom, sure.

MR. BAILOR: Your Honor, Dr. Brody has indicated to me that the presentation would probably be a little clearer and a little more effective if he would be able to borrow the portable mic and go near the screen to point out various things that he will show on the slides.

THE COURT: That's fine.

MR. BAILOR: All right. Could we get the portable mic?

(Pause)

THE WITNESS: Is it on right now?

THE COURT: Yes.

THE WITNESS: Is that all right, Your Honor?

THE COURT: Yes. Yes, sir, thank you.

THE WITNESS: May I stand here, then, Your Honor?

THE COURT: Wherever you're comfortable.

Q Okay. We have put on the screen ACC/FCR Exhibit 274. Can you explain what ACC/FCR 274 is?

A Sure. So, what I'd like to start with, Your Honor, is this diagram that really just acts as a map. It gives us a map for where the asbestos diseases develop.

So, for example, when you take a breath, the air

comes down this tube that we call the trachea or windpipe and you can feel the top of that in your Adams apple, that's the top of your trachea. You take a breath and the air comes down into these tubes that are called conducting airways because they conduct air down into the lung. Now, the diseases caused by asbestos as I say, we can use this diagram as a map. The first disease that you come to is actually found in the airways, in those conducting airways and that's called lung cancer. So, lung cancer develops in the walls of these tubes.

Typically, lung cancer develops in cigarette smokers. If a cigarette smoker is exposed to asbestos, they're much more likely to get the disease than if they smoke alone, or if they're exposed to asbestos alone. In fact, there's a synergy, there's an actual multiplication of the effect. So, it's not just adding the risk of getting the lung cancer from smoking and the risk from getting asbestos alone, you multiply that risk.

Out in the gas exchange area of the lung, which we'll look at more closely in a minute, is the disease asbestosis. Asbestosis, scar tissue in the lung, from inhaling asbestos.

MR. BERNICK: I'm sorry, could that be indicated again, with the pointer?

THE WITNESS: I'm sorry?

THE COURT: Could you --

MR. BERNICK: Show the little light, I missed the

little light.

THE WITNESS: Yes. That's in the gas exchange area round -- throughout the gas exchange area.

MR. BERNICK: I got it, I got it.

A This black line that runs around the outside of the lung is called the pleura, p-l-e-u-r-a. A very thin membrane, Saran Wrap thin membrane. Wraps around the outside the lungs, makes the lungs airtight, like balloons.

There are two diseases that develop at the pleura. One is another scar tissue disease, much like asbestosis but it's located just underneath that thin line, that pleura, and its called pleural plaque or pleural fibrosis. If it's pleural plaque, it's scarring in sort of concise patches. If it's pleural fibrosis, it's spread around the lung. But it's scar tissue, it's the result of an injury and just like asbestosis, it's scar tissue from inhaling asbestos.

And then there is a layer of cells that line the outside of the lung and those cells are called mesothelial cells.

THE COURT: I'm sorry, one second, please. Would you show me, please, on the diagram again?

THE WITNESS: Yes. There's a layer of cells that line the outside of the pleura, it's a complete sheet of cells lining the whole outside of the pleura, those cells are called mesothelial cells.

THE COURT: All right, thank you.

A If somebody has a mesothelioma, it means they have a cancer of those mesothelial cells.

Now, I use diagrams a lot when I lecture and I use various diagrams from textbooks, but it's good to be able to see what these things actually look like. So, if we go to the next slide, I'll be able to show you. So -- the lighting is not very good in here, but --

THE COURT: We can turn the lights down, if you like.

THE WITNESS: I mean, the slides will show up better, if it's not difficult to do. Ah, see that? Brilliant. Thank you, Your Honor.

THE COURT: All right. Can everybody -- can you see your notes, do you need to see your notes?

MR. BERNICK: I can see that the doctor was much younger then.

THE COURT: We were asking about notes.

THE WITNESS: We actually noticed.

MR. BAILOR: Weren't we all.

THE WITNESS: That's actually my purview as to whether I was younger or not.

A Actually, I had this microscope right up until Hurricane Katrina, so you can see how long I had this microscope and this is called an electron microscope and I can take a piece of tissue as small as a period at the end of a sentence, or as big

as this pointer I'm holding, put that tissue into this door right here, electrons come down from the top of the chamber. Inside this chamber is a vacuum, electrons come down, strike the tissue that I've put inside the chamber and an image then is recreated. An image of the surface of the sample is recreated in front of me. And just off of the screen is a camera, so I can take a permanent image of whatever it is I'm looking at.

So, if you back up to the previous slide for a second, and I cut a piece of tissue out of the lung and you can see the pleura running over the top, and you can see the airways going up into the lung, and I cut this out, and I put it into the microscope -- go ahead -- and I take a picture of it -- next slide -- this is what your lung looks like. Your lung looks like a sponge, you can see the airways going up into the lung, this is that very thin pleura that I was telling you about that runs over the surface of the lung and now, of course, it's cut across so you can see what it looks like inside. When you take a breath, the air comes into these airways, spreads out into the small airspaces that we have and this is where we exchange oxygen and carbon dioxide. I'll talk about that just a bit more in a minute.

Lung cancer, again, develops in the walls of these tubes, asbestosis out here in the gas exchange area, pleural plaque, pleural fibrosis, just under the pleura, and

mesothelioma out here on the surface and the mesothelial cells that line the outside of the lung.

Now, I'd like to spend just a minute talking about a couple of the defense mechanisms that we have. We all walk around the streets of Pittsburgh or wherever we are, we face bacteria, pollen grain, various kinds of dusts, a few asbestos fibers in the air and we have to clear those from the lung. So, we have a very effective set of defense mechanisms that help us do that, and I'll show you a little bit about them.

Our defense mechanisms really start with these nose hairs that we have and the moisture in the back of our throats, that's all part of the defense against inhaled particles. If we look at the surface of our airways, anywhere along the surface of our airways, and I focus the microscope anywhere along our airways, and I'm going to fill the screen with what's in the red spot. So, in other words, I'm going to -- I didn't ask you to do that, would you back up for a second. Thanks for anticipating, though.

So, what I want you to know is that the screen is going to be filled with what's in the red spot, and it could be anywhere along any of our conducting airways. Okay, so thank you, please go to the next slide. So, I'm filling the screen now and you see this says human bronchial and a bronchial is a small airway and our airways are lined by these little hairlike structures, they're not hairs at all, they're extensions of the

cell surface and these little hairs are called cilia and they're constantly beating in a wavelike synchronous fashion so that if something lands on the surface of our cilia, it'll get swept up to our mouth where we can swallow it or spit it out.

Now, notice that there are some cells here that do not have cilia and those cells make mucus, and unless you have a cold or you're a heavy smoker, or you have some airway irritation like I do from something, you don't think much about mucus, but your airways are constantly making mucus, the cilia are constantly sweeping it up to your mouth where you can swallow it or spit it out.

Now, there's a size marker in the lower right hand corner, the microscope is always telling us how big and small these things are. So, this bar says 10, with a little sign there that means microns, so I want you to understand how big a micron is, because at magnifications of thousands of times, it's easy to see 10 microns, but you need to understand how big a micron is. So, if I can do that for you, if you take your thumb and your forefinger and you make a little space that you can just barely see through with your naked eye, you've made just about one millimeter. Now, if you take that millimeter and divide it one thousand times, you've made one thousands microns. Okay? So, that means you can fit one thousand microns into a millimeter and you can just barely see one

thousand microns. Now, that means, with your naked eye you can't see 10 microns, you need a microscope to, of course, magnify it many thousands of times.

So, whenever we have an electron micrograph like this and we want to know how big or small something is, we take in our minds eye, we take this little bar and stand it up next to a cilium and you will see that the cilia are about eight microns long. And that's a typical human cilium and that's a typical rat cilium. Our cilia and the cilia of rats and mice and guinea pigs and horses are all working the same and doing the same thing.

And I also ought to point out now that if a rat or a mouse were running about around here, you'd be doing exactly what you and I are doing, inhaling and exhaling the room air, using exactly these same structures that I'm talking about.

Okay. If you'd back up for a second please. I'm going to now go past this structure of mucus and cilia and all of our airways are very much the same, we call that the muco -- for mucus -- ciliary escalator, because it escalates things up to our mouth. So, that whole defense combination we call the mucociliary escalator. So, I'm going to go back past the mucociliary escalator, out into the gas exchange area of the lung, because many fibers go right past the escalator and land out here in the gas exchange area of the lung, so I want to show you the cells that interact with the fibers.

When I started my work, we knew that asbestos caused all these diseases. I've told you I worked with Dr. Wagner, but we didn't know answers to simple questions like, well, where did the fibers go? We know they go in the lung but where in the lung do they go, where are they deposited? And then how do the fibers interact with the various cells of the lung, and then when they interact with them, how do they injure those cells of the lung? And those are the kinds of questions that I've been asking over the years. At that level.

Now, today, I'm working at the molecular level. That means the genetic level, the genes that drive these diseases. So, let's go, then, out into the gas exchange area and so, please, go ahead then, next slide, we'll go past the escalator, next slide, and out into the gas exchange. You can see this is the end of one of those airways, and we're moving now out into the gas exchange area and we're looking at a few of the hundreds of millions of airspaces that make up our lungs. Each one of these little airspaces, I like for instructional purposes, like to think of as individual rooms. Sort of like this room without a ceiling where it has the walls around it. It has a carpet lining the floor and we'll see that we have a kind of cell that lines our floors of our airspaces just like a carpet. Notice that when I opened some of these airspaces up -- and you maybe can see it better on your screen -- I opened up some little holes in the walls and you can see these little

holes in the walls, run through the walls, those are called capillaries; small blood vessels. All the blood in our bodies has to run through our lungs. This is where we're exchanging oxygen and carbon dioxide, right in these spaces. So you take a breath, the air from the room with about 20 percent oxygen goes flowing into these airspaces, the blood that's running through the walls picks up the oxygen from the room air and gives out the carbon dioxide that we've made in our muscles and our brain and we exhale. And, of course, that's going on all the time.

If you think about what's going on in the floors and the walls, if you were to cut through the floors and the walls you'd see the plumbing and you'd see the electricity and you'd see all the conduits running through the floors and the walls, and that's exactly the same as what's going on in the walls of these airspaces. The blood is running through these small channels, there are nerve endings running through, there is fluid flowing through these walls, and it's a constant process that is part of our normal function.

Now, of course, this is all normal, I have just a couple more slides in normal and then I'll show you what happens when asbestos gets in there.

So, the next slide, I'm going to take us into a single human airspace. And basically what you're going to be doing is kind of hanging over the room, no ceiling as I say,

and looking down on the carpet, on the rug. And the carpet in our lungs is made up of a series of, I guess sort of like carpet squares, different individual cells, they're not square of course, but let me show you what that looks like. So, now we're looking into a single human airspace. It could be any one of our airspaces. And I'm outlining for you here a single cell that makes up that carpet, but nature, as I say, doesn't make squares very well, it makes smooth surfaces, so this would be like a carpet oval next to another big oddly shaped cell and then another one over here and another one, so we have this patchwork of hundreds of millions of cells.

Now, just to give you a size perspective, from this side of the cell to this side of a cell, is about 40 microns. So, obviously, we can't see with our naked eye the individual cells that make up our airspaces. We have to have microscopes to be able to see them.

Now, these cells that are sitting here on the floor, all of these cells that line our airspaces are called epithelial cells, epithelial, e-p-i-t-h-e-l-i-a-l, epithelial cells cover surfaces. So, your skin is an epitheliam, we call it an epidermis. But our skin is an epitheliam, the cells that cover our airspaces are epithelial cells. So, we have these big flat epithelial cells that allow oxygen and carbon dioxide to move in and out of them as the blood transmits the gases from the blood that's flowing under. Then we have these

epithelial cells, and you can see these cells have sort of like bumps all over them and they're kind of short and squatty. These epithelial cells provide a repair mechanism. So, if the big flat cells get injured by asbestos, or infection or for any reason, these smaller cells with the bumps start to divide and take their place. So, we have this very effective repair mechanism in each of our airspaces.

Now, I have one more cell to show you that's normal and that cell is called a macrophage, m-a-c-r-o-p-h-a-g-e, one word, macrophage; "macro" means big and "phage" means eater. These cells are the big eaters of the lung. They patrol our airspace surfaces. We humans have, and rats and mice have about one or two of these macrophages sitting in our airspaces under normal conditions. Let's see what they look like. I'm going to focus the microscope right down on the carpet and remember the cell with the bumps all over it -- go ahead to the next slide -- and now there's the cell with the bumps all over it. So, you can see that I've magnified this again, here's the carpet down here, and now there are two other actors. There's this cell that's kind of ruffled and not going anywhere, then there's this cell with the tail end and a front end and I know it's going in this direction because it has these, what are called false feed or pseudo pods sticking out in front.

Now, I caught this cell in the act when it was going after this pollen grain right here. Now, this airspace once

belonged to somebody who was killed in a motorcycle accident. I was on the medical examiner's autopsy call and I went in to prepare this person's lung so it could be studied with an electron microscope and as I was going from airspace to airspace and taking various pictures, I saw these cells, these macrophages, guarding, essentially, our airspace surfaces. We don't want any kind of toxic particles on our gas exchange surfaces. So, these cells can detect the presence, through chemical signals, the presence of foreign agents. In my laboratory we discovered the chemical signal that attracts macrophages to asbestos fibers. And I'll show you that the macrophages are constantly trying to clean up the asbestos that lands down here on the airspace surfaces.

And so you've seen all the cells, now you need to see it to understand what happens when asbestos gets into the lung and so, therefore, we can go to the next slide and talk about asbestos.

Now, you can see this says chrysotile asbestos fiber bundle. And it's a bundle because that means that there are a lot of fibers stuck together, in a bundle. And let's look at the size marker so you get a feel for that. Down in the lower right-hand corner, this little marker right here, represents 1.0 microns. So, this is a one micron bar and you can see one micron pretty easily because I've magnified it 4,300 times. So, it's easy to see one micron.

Now, I'm showing you chrysotile, but I mean, I've used amosite, chrysitolite, chrysotile in my work, I use mostly chrysotile because it's what's used most in the world. It's about 90, 95 percent of the world's use has been chrysotile. All of the asbestos varieties cause all of the asbestos-induced diseases.

When I started working with Dr. Wagner, he was using chrysotile in his model systems and so -- and he showed me the design of the systems to be used to make the aerosols for the animal experiments. So, I basically continued that process right through to today. But be sure we understand, I've used amosite and chrysitolite in my work as well, but I'm going to show you what's going on using chrysotile as an example.

Now, chrysotile comes in an infinite variety of shapes and sizes and you can see that there are some short fibers and long fibers. If you were to put the micron bar on this fiber, it would probably be about seven or eight microns long. While these fibers can go off endlessly, hundreds of microns long. Some of these fibers are straight and some are curly and some are thin, extraordinarily thin. You can see this one passing right through the micron bar, is one-tenth of one micron. So, my point is, that there is an infinite variety of shapes and sizes and it's constantly changing. As the fibers fracture off of the bundles, they become smaller and smaller.

Now, if all of the fibers were to remain bound in a bundle, and the light were just right and it were floating by, it would look like a speck of dust, these bundles. If it all remained as a bundle, it wouldn't get past the conducting airways, too big. If you can see it with your naked eye, it's too big to get past the conducting airways. It's these fibers that are fracturing off of the bundles, those are the ones that are getting down into the areas of the lung where the asbestos diseases develop.

And what I'm going to show you in the next couple of slides are some slides from a couple of experiments of the many hundreds that I've done over the years, where I've taken rats and exposed them for a very brief time and when I say brief, I mean I put them in chambers about six feet high, four feet wide, and these are exposure chambers in which the animals are placed in cages in the chamber, a generator, an asbestos generator at the top of the chamber makes it very dusty inside the chamber. The animals then can inhale the dust for an hour, for days, or weeks or months, whatever I decide the time should be. I can expose them for a single hour and take them out of the chamber right after that, give the animals an overdose of anesthetic, of course, the animals don't wake up from that, and then I can show you exactly where fibers land in the first hour of exposure.

I can take another group of animals and look at them

hours later, days later, weeks later or months later, and again that's what a number of my papers are all about, is explaining this process. I wrote a chapter in a book called, "A Month in the Life of an Inhaled Asbestos Fiber". So, I mean it's tongue-in-cheek, but that's how you get to follow this process. You, obviously, cannot do that in people.

By the time people come to the clinic it's typically decades after they've been exposed, and so you don't get to follow the process in people, you have to follow it in the accepted animal models. And when I say accepted animal models, that's an important point because the work that I've described is in the open medical literature, it's been vetted by my colleagues and my peers and it passes the muster of the National Institutes of Health because all of my work is supported by the National Institutes of Health to do this work on understanding the disease sufficiently to develop treatments. There are no effective treatments for any of the asbestos-related diseases.

So, let's go to the next slide and look at one of these model systems that I've been using. Now, this is the lung of a rat. This is the end of the airway where it opens into the gas exchange area, and this, of course, is one of millions of these spots around the lung. I'm just showing you one for this purpose. And I'm going to focus the microscope right on this spot, immediately after a single hour of

exposure. Now, it's not my goal to bring these animals to the clinic. If I keep exposing these animals for months and years, they will eventually come to the clinic, all of them will get asbestosis, some of them will get lung cancer and fewer of them will get mesothelioma. That's been shown, that's been written over and over again. That's not my goal. I couldn't get funded to do that. What I had to do, and what I've done, is sort out the processes at the cellular and molecular level, the genetic level.

But, let's start by looking at what happens and then we'll go to the molecular level. So, I'm going to focus on this spot right here immediately after a single hour of exposure. So, I focus the microscope right here, take a picture -- next slide -- and there it is now. And remember the fibers that we saw sitting out on the dish, some of them were curly, some of them were straight, some of them were long, you can see a long fiber here. And this is a 10 micron bar. So, you can see this fiber is about 10 microns, this one is probably 20 microns. In other words, there is a size restricted group of fibers sitting down on the carpet. Remember that epithelial carpet I showed you in the human airspace. Same thing. This is now the rat, here are the capillaries, those tubes running underneath the carpet, and you can see the asbestos fibers sitting on the carpet.

Now, it's one of those things where we'd expose the

animals now and I'm working late at night in the microscope room and I'm going from airspace to airspace after this exposure and I said, I don't think anybody has ever seen this before, and what I'm saying is that what I saw was that the sum proportion of the fibers were being covered by the carpet cells. These cells, these epithelial cells that make up this carpet that covers all of our airspaces were actively coming up over the fibers and shoving them under the carpet.

Now, I'm telling you this in two minutes and, of course, it took me years to work all this out and prove it to my colleagues that this was happening and write a series of papers to prove that this was the case, but what's happening is, you can in fact see some of the fibers disappearing here, you can see others disappearing here, and if we go to the next slide, please, you'll see it even more dramatically, you can see there's a fiber here that's partway out, this of course is another animal, another experiment, there's an airspace here, another airspace. And there's a little bit of a fiber sticking out, there's a small bundle of fibers here. There are a group of fibers here. Some are covered by the epithelium and some you can see. There's a fiber here that's completely covered by the epithelium, all you can see is its electron shadow.

Now, perhaps you've noticed some of these structures here that look like doughnuts. These are your red blood cells. Red blood cells in rats and mice and people and every other air

breathing mammal, look like this, they look like doughnuts because they have a depression in the center, not a hole but a depression, and red blood cells from this side of the cell to this side of the cell are about five microns across. That's our red blood cells, rat, mice, again air breathing mammals, this is the size of the red blood cell that works. And so, it's been conserved through evolution and we can see those red blood cells here as they are marching through this capillary when I caught them flowing through the capillary and you can see another group of red blood cells flowing down this way, you can see that some of the fibers are being taken up and others are sitting here and the macrophages will be coming along and picking up these fibers, and the point now that I want to make is that this is a very dynamic process. All we can do is look at snapshots of this over time and fill in the spaces and work out the history.

What you have to recall, if you will, is that every time there is an exposure, every time a person inhales asbestos dust, some proportion of those fibers can land in the airways, those ciliated airways. Some proportion of those fibers get down on the carpet as everyone can see. Some proportion of those fibers get picked up by the carpet cells and shoved under the carpet. And then the story goes on because some proportion of those then can get to the pleura and cause mesothelioma.

So, the fibers that land in the airways are the ones

that contribute to participate in lung cancer formation. The fibers that you see here that are getting picked up and shoving under the carpet and that remain in that area under the carpet, those are the fibers that contribute to the development of asbestosis.

The fibers that are transported beyond these sites that I'm going to go to next, are the ones that contribute to mesothelioma. Okay? And so, every time there's an exposure, we have this proportional distribution of the fibers into the different compartments of the lung.

Now, I didn't tell you about another compartment, this compartment where these fibers are being transported to, underneath the carpet, I told you there's blood there, but I neglected to tell you that there's also what's called connective tissue there. Now, if you take your skin and you pinch it, hopefully, it'll pop back. It pops back because we have this elastic connective tissue that runs through our bodies, and gives us this flexibility and strength. This same elastic tissue runs through the walls of our airspaces.

So, when you take a breath the airspaces expand, when you release the breath, the elastic tissue brings that space back to its normal size.

In a person who has asbestosis, what happens is, these fibers that are being inserted under the carpet, are causing injury to the surrounding cells. These cells that you

see here are being injured. We've shown that, other investigators have demonstrated that, these cells are being injured. We know that because they start to divide, they leak, they allow fluids to come into the airspaces. When there's injury we make scar tissue. If you took asbestos and shoved it under your skin, you'd get a scar under your skin, because of the injury that's been produced by the asbestos fibers. Same thing in the walls of the airspaces. When there's injury in a particular area, there's a cell called a fibroblast, f-i-b-r-o-b-l-a-s-t. Fibroblasts make connective tissue. They make the normal connective tissue that we all need, and those cells make scar tissue in the face of injury.

So, when you have multiple exposures over time; now for asbestosis typically requires long term, occupational exposure, high levels of exposure, typically for decades, to bring a person to the clinic with asbestosis.

Asbestosis starts as soon as those fibers start making scar tissue, that's called microscopic asbestosis and that's what the animals are getting because I only give them a very brief exposure, it's enough for them to start getting a scar tissue response, but as I say I don't bring them to the end stage. But, a person who has asbestosis, that means there's been multiple microscopic injuries time after time after time, which our body responds to by making scar issue and all that adds up to this disease asbestosis.

And I'll show you what that looks like in a minute. But, the point I want to make here is, I want to get the fibers now, the proportion of fibers, out of this area to the pleura because it's the fibers that get to the pleura that, of course, cause mesothelioma.

Now, you see that the fibers are moving into what's called the fluid flow of the lung. You can see some of these fibers actually getting into the capillaries and I've demonstrated that, other investigators have shown that fibers get into the blood flow, you can actually see that happening here, but we have another kind of fluid flow in the lung. And that fluid flow in the lung is called lymph, l-y-m-p-h. And maybe you've heard -- I'm sure you've heard of the lymph nodes, and lymph fluids. Lymph is a clear fluid that runs head to toe in our bodies. Wherever blood flows there is lymph flowing around the blood flow. And there are two main functions for lymph flow. One is to carry cells of the immune system and that's why if you're getting a cold or you're fighting an infection you might feel your armpits swell, or the side of your neck get uncomfortable, because the lymph that's flowing there is being filtered by lymph nodes that are collecting inflammatory cells, cells that are fighting the infection and so, you feel it.

The other function is to help control pressure in our blood flow system. So, you can actually move fluids between

the blood and the lymph and that's a very important function.

But, going back to the first function of carrying cells of the immune system, not only does it carry cells of the immune system, but it turns out that lymph can carry asbestos fibers. Now, how do we know that?

We know that because some investigators, not me, but some investigators said, if in fact, there's asbestos in lymph fluid, then we should be able to find it in the lymph notes, these filters, and, in fact, that's what they did. They found lymph and let's -- if we go to the next slide, I'll show you what the lymph flow in the lung looks like. This is the pattern of lymph flow in the lung. And let me just explain, this is -- I don't know if you're familiar with Netter diagrams, Your Honor.

THE COURT: No.

THE WITNESS: Okay. So, this is Dr. Netter, you can see in the lower right-hand corner, Dr. Netter is an M.D., who fortunately for the rest of us decided to be a biomedical illustrator and what he's done is, he's given us atlases of the human body in health and disease, and I'll use a few Netter diagrams as I'm doing this. This is the first one and this is the lymphatic pathways in the lung and these very fine vessels that you see represent part of the lymphatic flow that ends at the pleura. You see this network that ends at the pleura and this network as a result of these very fine vessels that are

flowing out towards the pleura.

Now, if you'll back up to the previous slide, this is where the lymphatic flow in the lung starts, at the walls of the airspaces. In the walls of the airspaces, you can see there are capillaries, and then surrounding the capillaries is this fluid flow that collects into these vessels that you see in the next slide, please.

Okay. So, it's sort of like these are -- what Dr. Netter is showing you are the collecting tubes that carry the lymph to the pleura. Okay. Now, these little green blobs that you see surrounding the lung, those are some of the lymph nodes that we have. And as I say, investigators have found asbestos collecting in these lymph nodes because these are fibers that have collected in the lymph fluid and I showed you how those fibers first get there, because they land on the surface, get picked up by the lymph.

Interestingly enough, we have these lymph nodes in our peritoneal cavity which holds, of course, our stomach and our intestines, they're call mesenteric lymph nodes and investigators have found asbestos fibers in the mesenteric lymph nodes because they're part of this circulation. In other words, all of the lymphatic pathways are tied together in some way or another, just like our vascular, our blood system.

Okay. Now, what I'm going to do now is, again, let me remind you, so we have lung cancer in the walls of the

airways, we have asbestosis out here in the gas exchange and mesothelioma out here, and now there's one more diagram I want to show you regarding transport -- next slide please. So, this is a diagram that an investigator that I worked with for years, he was the chairman of the Pathology Department at Vermont and I knew him then, he put together a chapter to help explain this concept of migration of fibers to the pleura. So, he has this area now in diagram that I've been describing to you, he has a fiber coming in, inhaled, the fiber lands in the airspaces like I showed you, and then you see he's got this little fiber, just the end of it sticking out, and if you back up two more slides, remember this picture I took right here with the fibers sticking out, now the investigator didn't mean to draw this particular fiber, it just so happens we're talking about exactly the same thing. So, if you go ahead, then, to the one we had, and so now he's got the fiber here and he says lymphatic fiber transport to the pleura and he's got now the channel showing the fiber transport to the pleura and he's showing a normal mesothelium, that means there's a single layer of cells and then he has these various reactions that we're going to talk about in just a second, how these fibers then can cause mesothelioma out here.

Okay. Next slide. Okay -- skip that one, please. Okay. So, I told you the macrophages are trying to pick up whatever lands there, you can see there are one, two, three,

four, five macrophages, they're sharing fibers with one another, this is a 10 micron bar and this is a constant. I mean, we've done studies where macrophages have been collected from the lungs of workers decades after they've been exposed and you can still find macrophages with asbestos fibers in them, because the macrophages are constantly trying to clean out what doesn't belong in the lung. And these macrophages can actually live, not only in the airspace, but in the spaces underneath the epithelium, they move through that connective tissue space, pick up fibers, carry the fibers up and out of the lung.

Okay, next. Now, this is the clinical picture, sort of the end stage, asbestos and pleural fibrosis and I just wanted to show you that for a minute. So, this is decades after long-term occupational exposure, such that you can see the scar tissue, this white material in the background, you can see the scar tissue with your naked eye. If I took a little piece of lung up here where you cannot see the scar tissue with your naked eye and put it under the microscope, I'd show you scar tissue that you couldn't even see with an X-ray. But the point is, there are all different levels of scar tissue and it's happening in varying points around the body, at different times and then finally it can bring some people to the clinic with clinical asbestosis and pleural fibrosis. Now, you see that means there's also -- this is not cancer out here at the

pleura, this is scar tissue at the pleura.

Okay, next. Let's skip this one, it's the same thing. Okay. Now, this is lung cancer. And, this is, again, a Netter diagram. And this is the cancer and it is confined at this point mostly to the central regions of the lung which is where they start, because they start as I told you in the walls of the airspaces -- I'm sorry, the airways, the conducting airways.

Now, we always talk about target cells for disease. Every disease has a target cell. The target cell for asbestosis is the fibroblast, that cell that makes scar tissue. That's a pretty simple concept to understand. If that cell is surrounded by injury or it has injured itself, it makes scar tissue; asbestosis, if it's caused by asbestos.

The target cell for lung cancer are those cells that make up the mucociliary escalator. Now, you saw them early on this morning, I showed you the cells that make up the mucociliary escalator.

The cells that make mucus cells are the target cell for lung cancer, and I'm going to explain a bit more about that in a minute. The target cell for mesothelioma is out here on the surface of the pleura. Now, this is a normal pleura, this is the way a pleura should look, thin, shiny, Saran Wrap thin, to be moist, it has to be moist so that when a person breathes, when we breathe it rubs against the inside of our chest cavity

where there's another layer of pleura just inside of our ribs, and as long as everything is fine we don't pay any attention to that because it's moist and it's lubricated and that is what is supposed to be going on all the time. And as I say, this is a normal pleura. Okay, so this cancer developed from and I'm going to say now a single cell, and I'll explain how that works in just a minute.

All right. Let's go to the next slide. Now, this is a mesothelioma. This is the Netter diagram. You can actually see Dr. Netter's name kind of faded over here. Now, here the lung is quite normal, and that's not at all unusual in mesotheliomas, and I'll explain that in a second. But, here you can see the pleura is dramatically thickened, and now it's dramatically thickened with cancer cells. And the reason I say it's not unusual to see a mesothelioma with a normal lung -- with a normal appearing lung is, because many cases of mesothelioma develop from very brief exposures, low dose exposures, and so you don't see a lot of other asbestos-induced diseases at the same time.

You may or may not, I mean, you know, if a -- you know, these are all dose-response diseases. The more one is exposed, the more likely they are to get a disease. If a person's in a setting where they can get asbestosis, they're more likely to get mesothelioma, but that doesn't mean if you don't have these other diseases, you cannot get mesothelioma

from asbestos. That's not true.

Okay, so now what I'm going to do for the rest of the time -- and I have like five or six more slides -- is I want to explain how asbestos acts as a carcinogen, a cancer causing agent, and this is true both for lung cancer and mesothelioma. The concepts are the same. The target cell is different. The concept is the same.

Okay. Let's go to the next slide, please, and I'll do that. Now, this is a -- the cover of a proceedings of a meeting that I was at a few years ago. The topic of the meeting was how fibers cause cancer, carcino, cancer, genesis, formation, carcinogenesis; big word for cancer formation. As I say, I gave a talk about some of the stuff I've been telling you about today, and I showed you that cells can pick up fibers. You already saw that. But we haven't talked about and you cannot talk about carcinogenesis unless you talk about the molecular aspects, because that means your genes. Cancer is a genetic disease, so I'm going to give you the simplest definition of cancer, and then I'll come back and explain.

Cancer is the loss of control of cell growth. Let me say it again, so we'd be sure we have it. Cancer is the loss of control of cell growth. If I took a piece of your skin and I put it under a microscope, I can predict that about ten percent of your skin cells are growing, because you're always losing skin scales, and you have to replace them all the time.

So you have about ten percent of your skin cells are growing to replace -- normal replacement.

Your GI tract. You're always moving things through. We're replacing about 40 -- 50 percent of our cells at any given point in time in our GI tract; normal replacement. Our lung and our liver, one percent, a very low background rate of replacement. We don't expect to replace a lot of our lung and our liver cells at a rapid rate normally.

If you fall down and scrape your skin away, you'll get a very rapid replacement of that skin. Forty percent or so -- or 30 percent of those cells around the wound start to divide to make new skin cells, then when the wound is closed, they go back to the normal rate. That's all -- everything I've told you about cell growth, that's all normal. That's what's supposed to be happening.

Humans have about 20,000 or so genes. We have about 20,000 genes, and you can see what a few of them do -- a few of our genes do. You just look at different hair color, eye color, skin color, stature, all those things that are controlled by genes. You can see that that's just what a few of our genes do. Most of our genes -- most of our gene products you don't get to see. We make liver enzymes all the time. We're making various enzymes to protect ourselves against oxygen radicals. We have a whole set of genes making this fantastic waterproof skin that we have. So we have a

large series of genes and, of course, a lot of functions. All of our bodily functions are controlled by genes including cell growth.

So we have about 100 of those 20,000 some odd genes -- 100 of them are dedicated to controlling cell growth. Some of them have pretty obvious names like tumor suppressor genes. And I'll say a little bit more about those in a minute. We have a whole series of growth control genes, as I say, about 100 of them. And I told you that cancer is the loss of the control of cell growth.

Cancer develops when there are errors or mistakes in a set of growth control genes. Okay? So cancer is a loss of control of cell growth. Cancer develops when there are errors, mutations, mistakes, whatever you want to call them, in a set of genes -- not one gene, not two, not -- three is not enough. Four is probably not enough. I wish I could tell you exactly how many it takes, and I wish I could tell you just which ones it takes, and that, I'll tell you now, is one of the biggest problems we have in cancer biology today is the fact that in any given individual it takes a different set of genes to be injured for that person to get a cancer. That's why there's no set standard I can give you for how many asbestos fibers it takes, how many separate errors it takes. Nobody can do that, because it's different for different people.

What we do know is that asbestos is a carcinogen.

That means it's a cancer causing agent. That means it can cause errors in genes that control cell growth. It also -- it's a complete carcinogen. You don't need any other agents. Now, it happens to work very well, unfortunately, with cigarette smoke, which has a whole other bunch of carcinogens in it, so there it can be a co-carcinogen, but asbestos in forming mesothelioma is a complete carcinogen. You don't need anything else.

Okay, so in the rest of the time today I want to show you how asbestos acts as a carcinogen, as a cancer causing agent. And one of the ways we do that is by taking cells out of the body. We can take cells out of humans, animals, put them in a dish, give them the right nutrients, and those cells will grow and multiply. You get millions of cells in a dish, and then you can add the carcinogens or the agent you want to test. You can just add to those cells and see what happens and follow the molecular biology of those cells.

On the cover of this proceedings there are two cells, and you can see there's one cell here, and you can see there's another cell over here. And we've added fibers to these cells, and you can see that there's a long fiber and some short fibers, and those fibers are collected around the center circle in the cell. That center circle is called the nucleus, and the nucleus of our cells contains all of our DNA. DNA, of course, means our genetic material.

Notice how the fibers are excluded from the nucleus, and that's a good thing, of course, because our DNA is in the nucleus. We have this nuclear membrane. You can actually see this sort of bulge around the outside of the nucleus. That nuclear membrane protects our DNA, and that, as I say, is a good thing. That's an important defense mechanism that we have.

One of the things we've known for a long time in biology is that when cells divide, they lose that nuclear protective membrane. And, in fact, we know that when a cell is dividing, it's more likely to become a cancer cell. So we asked in my laboratory what would happen if we add asbestos when the cells are dividing. And I'll show you the results of that, but let me show you just a little bit about normal cell division first. We go to the next slide.

So this is cell division without any kinds of fibers in it, and let me show you what happens when cells divide normally. So here are three cells; one, two, three. The two cells on the outside are not dividing. The nucleus is intact. All the DNA has been stained blue, so you can see it. The cell in the center has received a signal to divide. Now, that could mean a number of different things. It could be the cell next to the wound where the skin's been scraped away, and your serum starts leaking in there, and all kinds of growth factors are in our serum to control cell growth, and the cells around the

wound start to grow, or these cells are in a dish, so I've added a growth hormone.

However -- whatever the reason is, these -- this cell's dividing, and I know that, because all of the DNA is condensed into these white threads called chromosomes. Chromosomes are bands of condensed DNA. Now, the whole object here is to make two cells just like the original. Scrape your skin away. You want cells to grow back just like the ones that got scraped away. The only way you can do that is by making perfect copies of all of your genes. So, let me show you what your chromosomes look like. Next slide.

We have 23 -- humans have 23 pairs of chromosomes. You got one chromosome from your mother, one from your father. Notice that there are light and dark bands on each of the chromosomes. Those light and dark bands are where your genes are. Now, some of those bands may have a thousand genes crammed in there. Some of them may have just a few. Each of our genes must be on the correct chromosome in the correct place on that chromosome. No mixing and matching allowed for the distribution of our genes on our chromosomes. So when I say you have to make perfect copy, when I say you have to reproduce your genes, I'm not kidding. That's exactly what we have to do.

Now, in the next slide we finish this normal cell division where here the cell has received a signal to divide.

You can see the chromosomes forming. They're stained purple. They have collected, and here they are undergoing faithful reproduction. If the cells undergo faithful reproduction, you'll get two what are called daughter cells just like the original. That's what you hope for every time.

Now, let's go to the next slide and see what happens when you have a carcinogen in the mix here. Now, this is from -- this particular experiment is from a paper that was published from my laboratory. And then in the next one I'll show you another example. Over here in Panel A -- now, again, you know, I'm showing you these one or two cells at a time, but we're talking about these going on in millions of cells around the dish. We're just picturing a couple of them for examples.

Over here in Panel A you can see a cell that's dividing. You can see half of the chromosomes gone to one side, half to the other, and that's exactly what you'd hope for to get to new daughter cells. Over here in Panel A you can see there are fibers. There's a long fiber, longer. There's a -- so from this side of the cell to this side of the cell is about 40 microns, so these are 30 micron fibers. This is a 5 micron fiber. Most of the DNA is moved to its respective location, but some of the DNA is bound to the surface of the fibers.

Now, that results in a condition called aneuploidy as you see in the upper right-hand corner here. Aneuploidy means abnormal chromosome separation. Let's look at one more example

of that, and this is my next to the last slide. Next slide. And now these are mesothelial cells, and over here on the right is a mesothelial cell with no fibers after the chromosomes have moved to one side, half to the other again. That's just what you'd hope for. Over here there are two cells, and there's a chrysotile asbestos fiber that is spanning the two cells and has bound some DNA. And again this is now aneuploidy produced in mesothelial cells by chrysotile.

Okay. The point here is that this is DNA damage. This is not cancer. Aneuploid cells are not cancer cells. Aneuploidy opens the door. Aneuploid cells are more likely to become cancer cells. When I say it opens the door, you have to open many doors before a cancer develops. So what I'm talking about now is, in a person who has a cancer what we know is that you must have a series of genetic errors over time. And let me just say this one more thing about genetic damage and then I'll summarize this whole thing of cancer.

Asbestos acts as a carcinogen in two major ways. One is that it binds DNA, and you can see that. I just showed you that in two slides. And so now here's some DNA that's not in its respective position on the chromosome. Now, if that can be passed on to the daughter cells, that opens the door a little wider. In other words, if this aneuploid cell can divide and pass on the error, the door opens a little wider, particularly if the genes that are on this DNA are growth control genes that

keep the standards of growth control where they should be. So asbestos acts as a binding agent for DNA.

Asbestos also generates oxygen radicals. Now, oxygen radicals are short-lived chemical compounds that are throughout our body and every living body all the time. We have a set of antioxidants that keep our oxygen molecules exactly at the levels they should be, and if they get a little higher, we start making more antioxidants to knock them down. But, oxygen radicals are known as very efficient ways -- the production of oxygen radicals is a very efficient way to produce genetic damage. Oxygen radicals damage DNA. They're the main way that cigarette smoke causes genetic damage, through the production of oxygen radicals. That's one of the main ways it does that. All the asbestos varieties generate oxygen radicals from their surfaces. So adjacent to these fibers -- the same time the DNA is binding to the surface of the fiber, the fiber can generate oxygen radicals and further damage the DNA.

Okay. Let's go to the final slide. All right. So I'm going to -- with this slide I want to summarize this issue of cancer formation. And I know you're familiar with this issue of latency; time from first exposure until the time the person comes to the clinic. What I want to -- hope to do here is have you understand what's happening related to cancer formation in that latency. With asbestosis it's actually pretty easy to understand what's going on in those decades of

latency. What all that means is you get this micro-injury after micro-injury wherever a fiber hits up against the cell, causes membrane damage. The fibroblast responds by making scar tissue over and over and over with what -- new fibers are coming in as well as the fibers that have been left in the lung as a legacy of the exposure. All those -- that's going on all the time.

For lung cancer and for mesothelioma, different story, and let me try to explain that here. So here's a layer of cells -- single layer --

THE COURT: Wait. I'm sorry. Excuse me. I misunderstood. That was the asbestosis you were describing.

THE WITNESS: That's correct.

THE COURT: I'm sorry. Okay. Thank you.

THE WITNESS: Right, the micro-injuries that add up the scar responses.

THE COURT: Scar responses. Okay.

THE WITNESS: Yes.

THE COURT: Thank you.

A Right. Okay. Now, we're talking -- now we're back to cancer, and here's a single layer of cells. And this is what mesothelial cells look like, but if you just think of them as airway lining cells, same concept. Single layer of epithelial cells. And then the artist has given us these lightening bolts, and it says DNA damage. I mean lightening doesn't -- as

far as I know, it doesn't cause DNA damage. It just means something from the environment. Something has come in from the environment, is able to reach the target cell.

Now, here the target cell is dividing, and I know that because you can see the chromosome. You don't see the individual chromosomes of a cell unless it's dividing, when those -- when the DNA is condensing into those chromosomes. Now, one -- and then he has the cell dividing, and he has one daughter cell going off in this upper left-hand direction and the cell is dying, and that is exactly what you hope for. One, the major fate of aneuploid cells is to die, and that is the main reason why most of us do not get cancer. We do not get cancer because our cells with genetic errors die. Now, they die, because we have -- and, of course, if they die, that means they can't pass on their genetic errors. That's a good thing. The main reason they die is because we have a set of genes that respond when there's DNA damage to send ourselves down that -- down what's called a suicide pathway. It's actually a cell death pathway that has evolved, and it's the same pathway in rodents and horses and monkeys and people that destroy cells with genetic errors.

Now, here's where I raise the issue of what happens if the genes that are damaged are the genes that control cell death, and now they can't send that cell down that pathway it's supposed to go? So that's one example of a genetic error that

wouldn't be in our favor.

Now, so then the artist also has -- and the other daughter cell with its chromosomes going in the other direction, and it's able to live. It's not going down that suicide pathway, and it's able to live. Now, you see the artist has this tumorigenesis or cancer formation, and he has this tumor with a whole bunch of oddly shaped cells going through these cell divisions. The time from this first daughter cell after DNA damage to the time the tumorigenesis forms, the division between this cell with the first damage and the development of the tumor is the latency period. So what you have to give me here for the rest of the discussion is this 30 to 50 or 60 years, whatever it might be, between the development of this first daughter cell and the cancer. So what's going on in that latency period?

So what you have to do is picture this cell with an error. See, it has DNA damage, and it's sitting there either in our airway or the surface of the pleura with an error, and it gets hit again. Another carcinogen, another asbestos fiber hits that cell again, gets a second error, and it starts to divide; two cells, four cells, six cells. Four of them go down the suicide pathway, but two of them are still sitting there with two errors. Okay? Those cells can divide. Our cells divide every 35/40 days or so.

Cells divide again, two cells, four cells, eight

cells. Most of them die, because they have errors. Now you have several cells sitting out on the surface, it's another error however many years later. It could be ten years later. It gets hit again. It gets another error. One of those cells or another of those cells gets another error. Now it's got four separate genetic errors, and it starts to divide; two cells, four cells, eight cells, 16. Thirteen or a dozen of them get recognized by our immune system, which is very good at picking up potential cancer cells, kills those cells, but you still have a bunch of cells -- now they're at various places around the pleura or in the airway.

Now, fast forward to 35 or 40 years, there's a cell which was the progeny of one of those groups, and it's sitting there now, one cell with six or seven errors. It gets another one. It gets an eighth separate genetic error, which for that cell in that person was sufficient to produce a cancer, and it starts to grow, and that's what the artist is showing you here. That's why he's made them all the same color, is because they all came from that same cell with a set of genetic errors which for that person was sufficient to allow the cancer to develop.

And the next person, he might have twice as many genetic errors and never get a cancer, just because it's a different set of errors or because that person's defense mechanisms were that much better. And then finally this grows out and brings that person to the clinic. Those are all the

slides I have, Your Honor.

THE COURT: Thank you.

(Pause)

BY MR. BAILOR:

Q Doctor, could you tell the Court if you had a block of asbestos, say chrysotile, the size of a sugar cube, how many fibers would that be?

A Oh, well, you know, I can't tell you exactly. It really depends on how tightly packed it is, but you could probably easily get a billion or so fibers into a sugar cube if they were packed.

Q Is all of your testimony that you gave here today so far based on reliable, widely accepted scientific principles?

A Yes.

Q And it's based on your own personal life's research?

A Well, most of it is, sure. I obviously draw from others as well, which is -- a scientist must do.

MR. BAILOR: Thank you very much, doctor. I'll pass the witness.

THE WITNESS: You're welcome.

THE COURT: Mr. Mullady, does the FCR have questions?

MR. MULLADY: No, Your Honor.

THE COURT: All right. Shall we take a ten-minute recess, and then we'll start on cross examination?

MR. BERNICK: Sure.

THE COURT: All right. We'll be in recess for ten minutes.

(Recess)

THE COURT: Please be seated.

(Pause)

THE COURT: Mr. Bailor.

MR. BAILOR: Your Honor, I'd like to offer at this time Exhibit ACC/FCR-271 -- that's Dr. Brody's CV -- and ACC/FCR-274, the slides he used to explain his testimony.

MR. BERNICK: Yes, we would object to both of those. The CV does not come in under Rule 702, 703 is not independently admissible. It's simply foundation for his credentials, and, therefore, his testimony. It's not a piece of evidence itself and it contains much hearsay.

Secondly, with regard to the demonstratives, they came in. They were used for demonstrative purposes. There would've been 100 foundational questions regarding exactly what fiber types were involved in these different demonstrations, exactly what kinds of cells, who took the pictures, when they took the pictures. In order to establish the predicates that these are, in fact, photographs that meet the requirement for the admission of photographs, because that's what they are. So they're just at this point, unless there is some further foundation -- and at this point I would object to going back over the whole testimony to provide it. Until there's further

foundation, they cannot be admissible as photographs, which is what they are. They would only be admissible as demonstratives, because they aid the testimony of the witness.

THE COURT: You're not offering them for anything other than demonstratives.

MR. BAILOR: They're only offered as demonstratives. And with respect to the hearsay objection to his CV, the author of the CV is sitting on the witness stand.

THE COURT: Well, yes, he is. The document itself is not the substantive evidence. The witness' testimony is. Nonetheless, I -- what I believe I have been doing is accepting these simply for purposes of my own recollection when I get to looking at the evidence, not for purposes of substantive evidence. Of course, the curriculum vitae, just like the expert reports, are not the evidence, the witness' testimony is. So, in that sense, 271 is not admissible as a substantive document, but the witness has been very clear about his background, and the slides that you showed I think illustrate quite clearly what the witness' qualifications are. So in that sense 271 is cumulative and not -- doesn't really add anything to the witness' testimony, so I won't accept it as substantive evidence, but it will be available in the event that I need it for purposes of refreshing recollection. And I -- Mr. Bernick, I don't think you'd have an objection to use it for that purpose, would you?

MR. BERNICK: No, not at all.

MR. BAILOR: Your Honor, we would like 271 in as substantive.

THE COURT: It is not substantive evidence. It cannot be admitted for that purpose. If you want to ask the witness every question about every entry on it, you may do so, but it is not substantive evidence.

MR. BAILOR: I don't think we want to spend that time. Thank you, Your Honor.

THE COURT: With respect to 274, I will accept it as demonstrative and only for purposes of demonstrative evidence. Let me make a note, please.

MR. BERNICK: Thank you, Your Honor. Is my microphone on?

THE COURT: It is, yes.

MR. BERNICK: Okay. Thank you.

THE COURT: Just a minute though, please.

MR. BERNICK: Sure.

(Pause)

THE COURT: All right, Mr. Bernick, thank you.

MR. BERNICK: Thank you, Your Honor.

CROSS EXAMINATION

BY MR. BERNICK:

Q Good morning, doctor. I want to start out by asking you some general questions with respect to two diseases or two

kinds of disease that I believe that you've discussed; one is fibrosis and the other is cancer. And, actually, you talked about two different kinds of cancer. You talked about lung cancer, and you talked about mesothelioma, correct?
A Yes.
Q And would you agree with me that those are two different types of disease?
A Certainly.
Q Okay. And just to go through very, very briefly, some of the characteristics that you've highlighted that illustrate why they're different, I think you said that fibrosis has as a target cell the fibroblast?
A Correct.
Q And the function of the fibroblast in the body is basically to lay down fibrous tissue, correct?
A Right.
Q That's what they are. Right? They -- I'm sorry -- they assist in the process of creating that fibrous tissue, corrrect?
A Right.
Q Is it true that almost any kind of solid foreign body introduced into the human body that persists over time will ultimately be surrounded by fibroblasts and fibrous tissue?
A No question about it. Asbestos is very good at doing that. There are different degrees to which the fibroblast

responds, but what you say is true.

Q Is it also true that the reason the fibroblasts are recruited and the reason that that fibrous tissue is laid down is it's one of the body's defense mechanisms?

A That's right.

Q And is it also true that the system that's involved in that reaction by the body is the immune system?

A Well, it can be. I mean, the immune system can be involved in the -- in that process. Sure --

Q Well --

A -- not necessarily.

A Well, but there is no other system that recruits these cells in response to the inflammatory reaction that takes place when the body is introduced other than the immune system. It's the innate immune system, correct?

A No, that's not correct. Fibroblasts can produce structures that attract other fibroblasts, so I'm not sure where you got that. The only --

Q Well, what recruits the fibroblast to begin with to the site?

A Well, certainly, the immune system can be -- is involved. No, excuse me. The immune system is involved in many cases, but you said it's the only one, and I've corrected you. That's all.

Q Isn't it true that the innate immune system is involved in

each and every case where foreign material is introduced into the human body?

A You know, I don't know why you say each and every case, you know.

Q I'm sorry. My intentions and my motives are not really relevant. I just asked you a question. Isn't it true that in each and every case where a foreign material is introduced into the human body the immune system becomes activated?

A I just don't know that that's true. I mean, it may be, but I can't answer it with enough authority to agree. I just don't know.

Q Okay. You would agree with me though that this is defensive; that is, the walling off process?

A Correct.

Q And would you also agree with me that it is possible over time for this reaction to become stable or quiet?

A Sure.

Q Let's talk a little bit about cancer. I think you told us that in -- there are different target cells that are involved in cancer --

A Right.

Q -- correct?

A Right.

Q And we also know that when we have cancer, we have tumors that are formed, right?

A Right.

Q And is it true that with respect to cancers not every foreign material that's introduced into the body and persists causes cancer, correct?

A True.

Q It has to be carcinogenic?

A That's right.

Q Is it also true that -- I think you testified to this, and I found it a particularly clear statement as well as many of the other statements that you've made -- that cancer is characterized by not stability but by a loss of control over cell growth?

A That's fine.

Q Okay. Now, with these differences in mind, I want to talk a little bit about your own experience. Isn't it true that your research has focused on fibrosis?

A Largely, that's true, although my work also has focused on cell growth and cancer formation, but largely what you say is true.

Q In fact, isn't it true that the website that you have had at Tulane as recently as 2001, that that website focused specifically on your work concerning fibrosis and fibrogenesis?

A Probably, sure. .

Q Well, do you remember one way or the other?

A I -- you know, I'd have to go back and look, but I'm sure

it also stated my --

Q Excuse me. I just want to ask you whether you remember --

MR. BAILOR: Could we have the witness complete his answer?

MR. BERNICK: No, it's not responsive.

THE COURT: Pardon me. Address me not the witness, please. I think the answer first was a yes or no, and then yes, the witness may explain his answer. So, doctor, if you could answer yes or no, please, then you may explain your answer.

THE WITNESS: Sure.

A I don't remember everything it says. I'd have to read it, and it may very well have gone into my work with control of cell growth. But, I'm sure I did focus on fibrosis, because that's what I've been focusing on for most of my career.

Q I want to show you Exhibit 763 and ask whether this is a copy of your website at Tulane when you were there as Professor and Vice Chairman and Chief of Lung Biology?

A Yes.

Q And do we see that when it talks about your interests, you talk about research ongoing in the laboratories of the Lung Biology Program is focused on the biochemical and molecular mechanisms that mediate fibroproliferative lung disease caused by inhaling environmental agents. Do you see that?

A Sure.

Q And do you see you then go on to talk about what that proliferation is; that it's a scarring process, lung damage results and cellular proliferation, the two hallmarks ultimately of pulmonary fibrosis, correct?

A Absolutely, and along with these papers that are listed here which deal with tumor necrosis factor and control of cell growth. Sure.

Q Dr. Brody, isn't it true that you nowhere in your website hold yourself out as being an expert in carcinogensis?

A I don't hold myself out as an expert in anything on my website. This is the work that I do. That's all.

MR. BERNICK: Your Honor, again, I would ask that the witness try to be responsive to my questions. I asked him a very simple question. Does his website anywhere hold himself -- him out as being an expert in carcinogenesis?

THE COURT: That was responsive. He said it doesn't hold himself out as an expert in anything on the website.

THE WITNESS: Yes, thank you, Your Honor.

THE COURT: That was responsive.

THE WITNESS: Thank you, Your Honor.

MR. BERNICK: Well, it's not -- again, would you instruct the witness that it's not up to him to express appreciation to the Court either.

(Laughter)

THE COURT: That is a given, sir. Thank you.